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Smoking as a risk factor in periodontal diseases

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Purpose

The purpose of this Clinical Update is to provide the clinician with a general overview of the relationship and impact of smoking on periodontal diseases. The following main topics will be addressed: 1) the effect of tobacco smoking on the prevalence and severity of periodontal diseases; 2) the effect of smoking on the response to therapy; 3) possible mechanisms of periodontal disease progression in smokers; and, 4) the effects of tobacco cessation.

Introduction

Tobacco use, smoking in particular, is directly related to a variety of medical problems and appears to be one of the most significant risk factors in the development and progression of periodontal disease. Approximately 25% of the adult population smoke cigarettes. Although this percentage has been declining since the 1970's, the rate of decline is less among women and certain minorities. Tobacco use has grown more popular among youth (1). The use of smokeless tobacco products and cigars may also affect periodontal health and is of concern, particularly among young males (2).

Smoking effects on the prevalence and severity of periodontal diseases

The Third National Health and Nutrition Examination Survey (NHANES III) is a multi-purpose health survey that was conducted from 1988 to 1994 by the National Center for Health Statistics of the Centers for Disease Control and Prevention. An epidemiologic survey of 13,650 civilian, non-institutionalized dentate adults (aged $\geq$18 years) who received a periodontal examination and provided data on their cigarette smoking habit, established an association between cigarette smoking and periodontitis. Data from this epidemiologic study revealed that 28% of the representative dentate population were current smokers and 23% were former smokers. The overall prevalence of periodontitis in this study population was 9%. Analysis of data suggested that current smokers were approximately 4 times as likely as persons who had never smoked to have periodontitis, after adjusting for age, gender, race/ethnicity, education and socioeconomic status. Among current smokers, there was a dose-response relationship between cigarettes smoked per day and the odds of periodontitis. Among former smokers, the odds of periodontitis declined as the number of years since quitting increased. The authors concluded that smoking is a major risk factor for periodontitis and may be responsible for more than half of periodontitis cases among adults in the United States. A large proportion of periodontal disease may be preventable through smoking prevention and cessation of cigarette smoking (3).

In a separate investigation, the Baltimore Longitudinal Study of Aging found that cigarette and cigar/pipe smokers had a higher prevalence of moderate and severe periodontitis, and a higher prevalence and extent of attachment loss and gingival recession than non-smokers. Smokers exhibited less gingival bleeding and higher numbers of missing teeth (4).

A 1993 study by Haber, et al., found that periodontitis is more prevalent and severe among current cigarette smokers than those who never smoked, as determined by measurements of pocket depth and attachment levels. Current smokers possessed a greater number of affected sites per subject and a higher proportion of deeper sites than non-smokers (5).

Effect of smoking on the response to periodontal therapy

Current smokers show less reduction in pocket depth and less gain in clinical attachment level compared to former smokers and non-smokers after nonsurgical mechanical debridement. Also of note was the persistence of the periodontal pathogens, *Porphyromonas gingivalis* and *Bacteroides forsythus*, in current smokers after scaling and root planing (6).

In a longitudinal comparison of therapies study, Kaldahl, et al., reported seven-year results from patients who were treated surgically, employing the Modified Widman Flap in one quadrant, and flap with osseous resection in another quadrant, versus non-surgical scaling and root planing or coronal scaling in the remaining quadrants. Overall, regardless of the treatment modality, current smokers consistently exhibited less probing depth reduction, less clinical attachment gain and more loss of horizontal attachment in the furcation area than former smokers and those who never smoked (7). Root coverage attempts utilizing thick free gingival grafts were adversely affected by heavy cigarette smoking (8). To date, there is no conclusive evidence implicating smoking as a factor limiting success in subepithelial connective tissue grafts.

Tonetti, et al., reported on results of guided tissue regeneration therapy utilizing non-resorbable barrier membranes in deep intrabony defects. At the one-year follow-up, smokers had gained significantly less probing attachment level than non-smokers (2.1 mm v. 5.2 mm) (9). Some studies have reported reduced success rates with implants in smokers. In one study, investigators found of the total 390 implants placed in smokers, 44 failures occurred by stage 2 for an 11% failure rate. This was significantly increased from the 4% failure rate observed in non-smokers (86/1804). Overall implant success rate for up to 6 years in function among these current smokers was 89% as compared to 95% in non-smokers (10). In 1992, Jones and Triplett reported on simultaneous grafting and implant placement. In their group, smokers accounted for 80% of all wound complications with dehiscence and infection the most common of complications. In total, smokers comprised only 33% of the study population, yet exhibited the majority of post-operative complications (11).

Possible mechanisms of periodontal disease progression in smokers

Although bacteria are the primary etiologic factor in periodontal disease, the patient's host response is a major determinant of disease susceptibility. The majority of the literature reports conflicting data with regards to differences in amounts of bacterial plaque and calculus and virulent types of the pathogenic flora in smokers versus non-smokers. In general, the focus on pathogenesis has been on the impaired or inappropriate host response. Smoking may lead to increased periodontal destruction by altering the host response via 2 main mechanisms: 1) impairment of the normal host response in neutralizing infection, and

2) alterations that result in destruction of the surrounding healthy periodontal tissues (12). Researchers have reported decreased or impaired neutrophil phagocytosis and/or chemotaxis in smokers as compared to non-smokers (13). Smokers appear to have decreased numbers of helper T-lymphocytes, which are critical in B cell function and subsequent antibody production resulting in an impaired response to various pathogens (14). Smoking has also been shown to cause neutrophils to be over-stimulated or impaired in the release of enzymes during the oxidative bursts causing excessive degradation of host tissues or perhaps ineffective antimicrobial function (15). It has been hypothesized that smoking has a deleterious effect on gingival blood flow. The initial work on the effects of nicotine demonstrated a decrease in gingival blood flow based on heat diffusion studies (16); however, later research using different methodology yielded contradictory results (17). Tobacco toxins may also modify the production of host-derived inflammatory mediators, which results in tissue destruction. These alterations in host response may not only adversely affect the progression of periodontitis, but may also diminish the reparative and regenerative potential of the periodontium.

Effects of tobacco cessation

In general, the periodontal status of former smokers is intermediate between that of those who never smoked and current smokers; and, although the effects of smoking on the periodontium cannot be reversed, there are significant benefits to tobacco cessation (5). Clinical investigators found that when comparing former smokers to current smokers and those who never smoked, former smokers (quit >1 yr) showed reduction in pocket depth and gain in attachment level comparable to non-smokers and significantly better than current smokers (6). Observations on the effect of smoking cessation on periodontal status and salivary components, suggest that the prevalence of pocket depth $\geq$ 4 mm, gingival suppuration, and loss of crestal bone were significantly lower, and that the salivary buffering capacity was significantly higher in subjects who had quit smoking as compared to current smokers (18). These data are encouraging evidence for clinicians and their patients, and emphasize the positive impact of smoking cessation on periodontal therapy response and future maintenance of the periodontium.

Current Navy guidelines mandate dental officers to provide the following intervention at each annual examination (19):

1. Inquire about the use of tobacco
2. Document the amount, duration and type of tobacco used
3. Inform the patient of the hazards of tobacco use
4. Inform the patient of the benefits of quitting
5 Inform the patient of the availability of tobacco cessation programs
6. Inform all pregnant tobacco users of the hazards of tobacco use to the developing fetus

Although smokers benefit from periodontal therapy, clinical improvements are less than those for non-smoking patients. Based on the evidence, tobacco use places the patient in a high risk category for periodontal diseases and intervention strategies should occur at each patient visit. The dental officer may use the patient dental and medical history, chief complaint, oral cancer screening examination, current dental examination, including the periodontal findings, to educate the patient about the hazards of tobacco use and the benefits of cessation. Appropriate referral to a tobacco cessation program complete with specific information capitalizes on the patient's desire to quit.

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